sis was observed in the group undergoing hyperventilation with 6% CO_2. It is concluded thal hemolysis is unrelated to mechanical action of hyperventilatroin and in due to alkalosis. the possible cause of hemo lysis and related litrature is discussed.

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INTRAUTERINE INFECTION. *

M. H. Karimi - Nejade. M. D. **

Pothways of fetal and early neonatal infection.

Review of the «AMNIOTIC INFECTION SYNDROME» in 150 autopsies of new and stillborn babies.

The risk of intrauterine infection occuring in the newborn infant was mentioned by KUSSNER (1877) and GEYL (1880) at the end of the last century (14).

SLEMONS (1915) reported the occurrence of bacteria in the sub-amniotic space near the attachment of the umbilical cord during prolo-nged labour (21).

DOUGLAS and STANDER (1943) have shown that mortality and morbidity of the newborn are directly related to the length of labour and this is on account of intrapartum infection (13-14).

In recent years obstetricians, paediatricians and Pathologists have recognized the problem of intrauterine infection and, among them WILIAM and KURT BENIRSCHKE have written much about this subject.

According to MULLER (1956) the foetus may be infected by the following routes:

1- Hematogenous spread via maternal blood.

2- Ascending amniotic infection in which vaginal bacteria reach the uterine cavity directly through the cervical canal.

3- Transdecidual spread, either on account of an exacerbation of an existing endometritis or because of an ascending infection occuring between the uterine wall and the memoranes.

4- Via the fallopian tubes.

From a practical view point the hematogenous spread which causes antenatal infection and the ascending amniotic infection which occurs during labour are the more important causes of intrauterine infection and early neonatal infective death (2-4-6-7-14-18-20).

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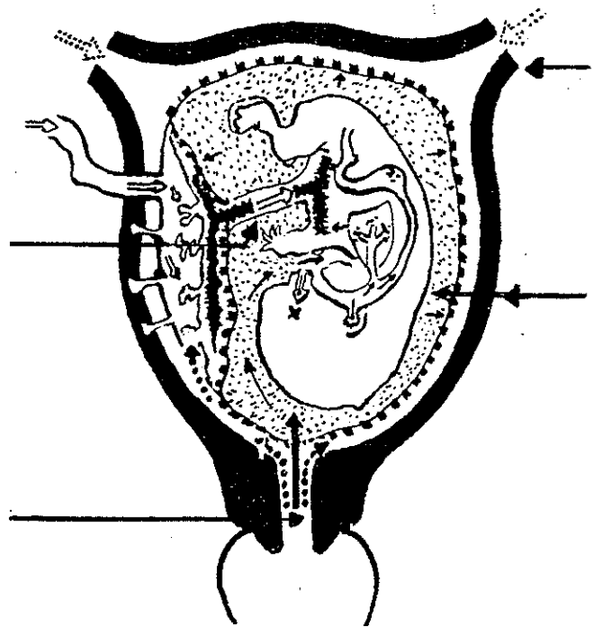


Figure I, The route of foetal contamination.
, Hematogenous spread
, Ascending amniotic infection
, Asending placento-foetal infection
, Via Fallopian tubes.

The way in which hematogenous spread occurs is not well known. It seems that the microorganism enters the intervillous space and then passes to the fetal circulation. However, intrapartum infection is more common and this is due to vaginal bacteria reaching the amniotic cavity even in the presence of intact placental membranes in some cases (4–8–12–16–10–21).

In the presence of amniotic infection the fetus is usually infected through the respiratory or gastrointestinal tract, the more severe infection being seen in the lungs (4–8–10).

The inflammatory reaction which seen in the umbilical cord, placenta, membranes, and fetus is called the « AMNIOTIC INFECTION SYNDROME» by william Blanc (1959)

Transdecidual and tubal spread occur only rarely (4–7–8–13–14–18–20).

The two more common means of spread, namely, hematogenous and ascending amniotic will be discussed in this paper.

I- Hematogenous route: this type of infection could occur during the whole pregnancy Some infections attach the zygote and some, such as toxoplasmosis snd cytomegalic inclusion, affect the fetus. (4–7–8)

Etiology: The following agents could infect the fetus: Viruses: herpes simplex, herpes zoster, variola, small pox, poliomyelitis, rubella, cytomegalic inclusion, viral hepatitis, mumps, mononucleosis, lymphogranuloma inguinale, influenza, psittacosis and coxachie.

Bacteria: The intestinal bacteria (such as coliform bacillus and enterococcus), vibrion faecalis, clostridium, pasteurella, listeria monocytogenes, mycobacterium (tuberculosis-lepra), spirochete (treponema pallidum and leptospira).

Fugi : Histoplamosis, coccidioidomycosis and candida albicans.

Protozoa : Toxoplasma gondi, plasmodium and trypanozoma.

Among the viruses rubella, poliomyelitis. influenza, coxachie and salivary gland infection and among the bacteria coliform bacillus, streptococcus, pneumococcus, listeria monocytogen, toxoplasma gondi, tuberculosis and treponema pallidum (of which the two later are so frequent today) are more important.

Pathology of placenta:

The way in which the pathogenic organism passes through the placenta is not yet clear and it is still not known if it is necessary to have a placental infection or not. In the cases examined the inflamation was situated in the intervillous space and merged with the adjacent villi. 8-18 In several cases of congenital tuberculosis the placenta was infected via the hematogenous route but the interesting thing was that the chorionic surface was infected direcly via the infected amniotic fluid. (8) In several cases which the author studied, apart from obvious placentitis, he could not see any pathological change except fetal maceration.

II- ASCENDINC AMNIOTIC INFECTION:

The most common route of fetal infection is the ascending amniotic one which has been known since the last century and which has been proved by many authors. (2-4-6-10)

The newborn's nasopharynx ususlly contains bacteria derived from tha mother's intestine. LARS ENGSTROM BORN IVEMARK in an experimental work containing 151 deliveries have shown the increase of these cases in which labour was prolonged more than 24 hours and in those cases where the membranes ruptured prematurely. (14)

Emmrich has examined the passage of bactaria through tne placental membranes. He showed the presence of unruptured membranes. 3

Hermstein (1930) studied the elasticity and permeability of membranes and showed that the chorion is more permeable than the amnion. Coagulase positive staphylococcus aureus passed through the chorion within a few hours but it took 6 hours to pass through the amnion. Aurelius and Engstrom studied different bacteria. They found that although the coliform baccillus had passed through the chorion within 14 hours it had not passed through the amnion within 24 hours.

They thought it was very unlikely that the bacteria could reach the amniotic cavity that there was a small rupture in the upper part 2-14

The important thing is a combination of ruptured membranes and uterine contractions which increases the risk of infection. (8-16-20)

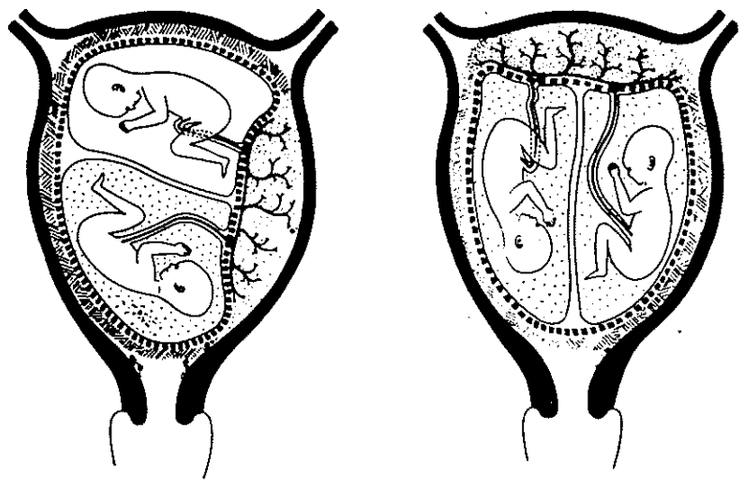
The other thing which shows clearly the importance of asceding amniotic infection is the study of twin babies and twin placentas. KURT BENIRSHKE studied 170 twin placentas and found there were 23 cases of placentitis as follows:

17 cases of first twin infection

6 cases of both twin, infection

There was no case of second twin placetal infection alone.

It is clear that the majority of twins are situated as in figure A and rarely as in figure B (In the latter circumistance the infection can reach both twins equally) If the infection was ascending, as we believe it is, the first twin's infection would be more common and this is compatible with Benirshke's findings. In the six cases of inflamation of both twins,



Figuer 2, A B

Situation of twins in the Uterus

this figure is reproduced from Benirochke's paper Ref. No. 4

placentas we should exclude two cases because in one there was no separating membrane (Monochorionic) and in the other the second twin was delivered a week later. Thus, there were only four cases of double placentitis. This is compatible with the idea of ascending amniotic infection.

The other thing which was important in Kurt Benirshke,s study was that there were 7 cases of monochorionic twins in which there were direct anastomosis between the two twins' circulation. In spite of this in only three of these cases were both placentas infected and in one of these there was a common ammiotic sac monochorionic monoamniotic). 4

There are different ideas about the length of labour and the length of time the mombranes have been ruptured. Generally, when rupture of the membranes has occured more than 6 hours before delivry and labour more than 24 hours it is considered to be a case of early rupturd and prolonged labour. (4-9-8-14-18-20)

It is obvious that the following factors are acting in the amniotic infection syndrome:

1- Premature rupture of the membranes with or without onset of labour.

2- Labour lasting more than 24 hours

3- Slow dilation of the cervix even in the presence of intact membranes

4- Internal manipulations

Other factors such as prematurity, uterine inertia, large baby and stillbirth predispose to infection.

Bacteriology: The most commonly found bacteria in the amniotic fluid are:

1- Coliform bacillus

2- Enterococcus

3- Coagulase positivve stapyhlecoccus aureus

4- Streptococcus faecalis

5- Staphylococcus albus

These bacteria are found also in the vaginal pool.

Gosselin has found the same bacteria in the heart blood of 17 cases of 117 examined cases in which these organisms were already present in the amniotic fluid.

Pathological findings:

Placentitis : placentitis which occurs owing ascending infection affects mostly the omnion and involves the chorion and placental tissue so william Nianc has suggested the amniotic smear as a tool for amniotic infection.

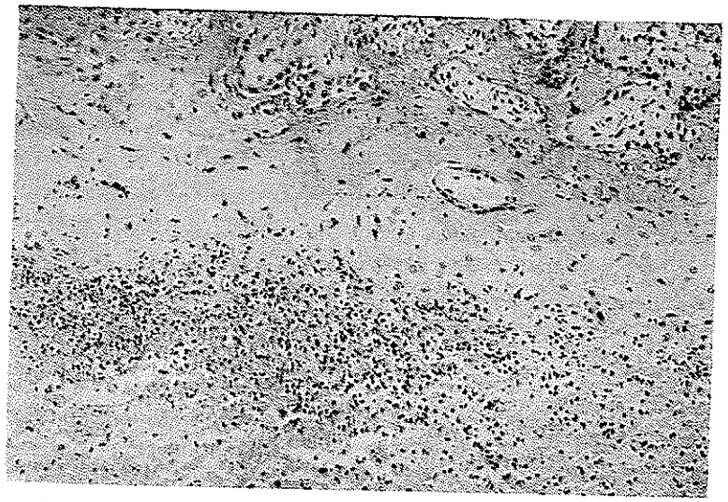


Figure 3, Placentitis. note the infiltration of polymorphonuclear cells beneath the amnion (within chorion). The placental villi are seen in the lower part.

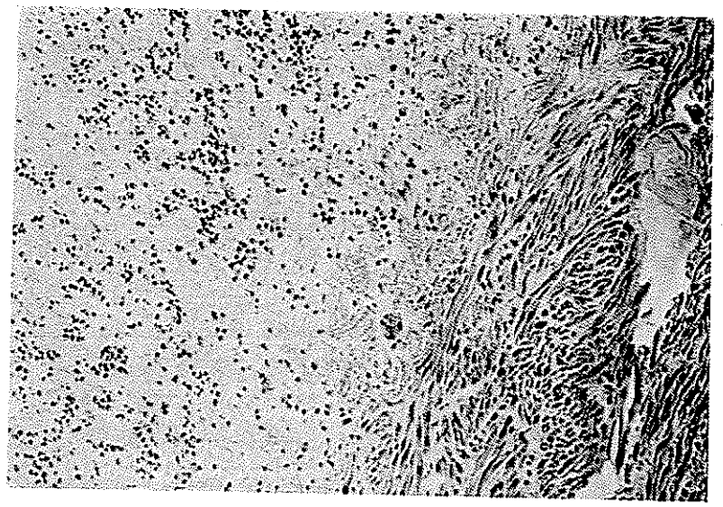


Figure 4, Intensive omphalitis. The polymorphonuclear leukocytes have infiltrated the warton jelly and lie between the muscular fiberes of the walls of vessels (Vasculitis).

Umbilical cord: Infection of unbilical cord is very common. It occurs as an inflammatory infiltration in the warton Jelly and around the vessels (vasculitis). It was seen in 10% of 1300 serially axamined placentas in which there were 12.7% cases of chorioamnionitis.

Kurt Benirschke advised immediate examination of the umbilical cord as an aid for early diagnosis of the amniotic infection syndrome. He suggested one should take a piece of umbilical cord 3 cm in length, fix it in a 10% formaiin solution and examine it by the frozen section method. (3-4-8-18)

Fetal infection:

All fetal organs can be infected e.g. otitis, congenital meningitis, interstitial nephritis, inflamation of the gastrointestinal tract and hepatitis (which may occur following omphalitis) and also septicemia. Neverthless the most severe and important pathological condition is intrauterine pneumonia.

Intrauterine pneumonia: In the severe case it causes intrauterine

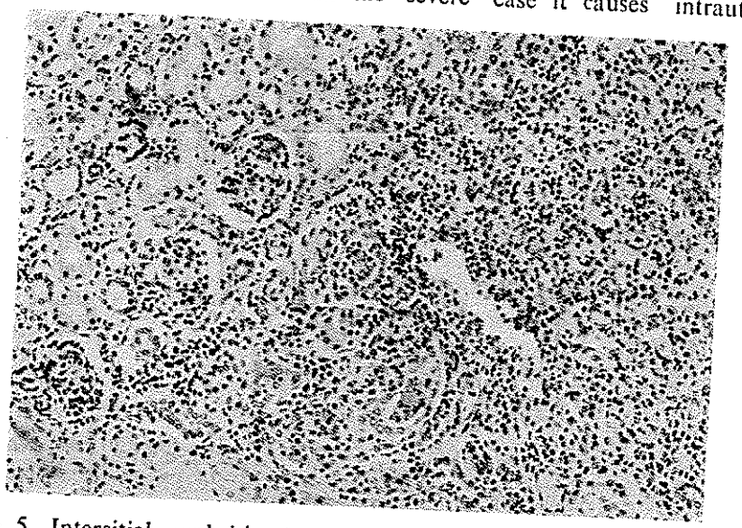


Figure 5, Intersitial nephritis. Note the infiltration of inflammatory cells around glomerulae & tubules.

death. In the less severe case the newborn child may expire immediately in a few instances or within the first three days after delivry.

The pathological appearenee of intrauterine pneumonia is usually charcteristic and quite different form postnatal bronchopneumonia. The clinical symtoms are similar. The prognosis is poor but is not fatal in all cases. (16-17-18-19-20-21-22)

Pathology: The lungs are in the consistency of liver. Infection

is there is no abscess formation and the respiratory tract contains no secretion.

Microscopic examination: In advanced affected. There are large number of polymorphonuclear leukocytes and occasionally monocytes in the alveoli and fibrin is scanty. The amniotic constituents are not abundant although in some cases, they are considerable; the later seems to be infected amniotic aspiration. (10_15_16_17_20)

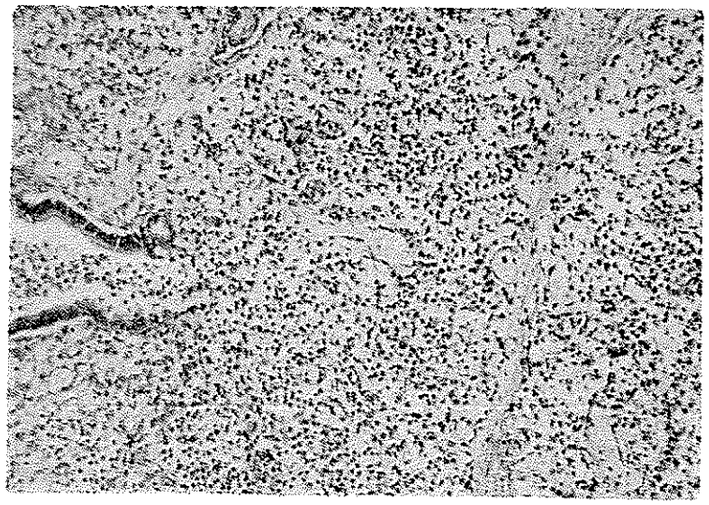


Figure 6, Intrauterine pneumonia. Note the tissue is uniformly affected. The alveoli and bronchial lumena are filled with inflammatory cells; mainly polymorphonuclear.

Occurance: Johnson and Myer found 19% - cases of pneumonia in 500 autopsies of new and stillborn children of which 13% showed this characteristic intrauterine pneumania.

Other authors found higher percentges of cases and it has become clear recently that better hygiene decreases these percentages to 6 or 10% of all autopsies of new and stillborn children.

According to most authors, pneumonia in the newboran is less common than massive amniotic and hyaline membrane; but in this study, as will be clear later, it is much more common. (15.20)

Pathogenesis: All factors which are responsible for amniotic infection are also responsible for intrauterine pneumonia on account of amniotic inhallation by the foetus.

Diagnosis: In the mother amniotic infection should be looked for in all cases in which amniotic infection is likely to occur, such as prolonged labour, premature rupture of the membranes and the presence of high maternal fever. 8_20

In the full term baby the history is important, delayed crying and breathing, tachypnoea and stertorous respiration may exist. Fever is usually present but is rarely predominant.

Physical symptoms: Bronchial breath sounds and ronchi are present. The infant may be flacid or spastic and, even convulsion may occaionally occur. Cyanosis and cardiac failure may be seen. Radiography does not help usually.

Immediate frozen section examination of the umbilical cord and cytological examination of the inner surface of the amnion are the best and safest tools for early diagnosis.

Prognosis: A severe infection ugually ends in the intrauterine death of the foetus while the less severe one results in alive born infant who dies from pneumonia, generally within the first few days of neonatal life inspite of reasonable treatment.

Treatment: Culture of respiratory tract secretion, fixation of the bacteria and performance of an antibiogram are essential so that the appropriate sensitive antibiotic be administered as soon as possible.

Prevention: Von Friesen gave the pregnant women with fever, prologed labour or inertia antibiotics. He decreased the mortality by about 50%. Other workers gave streptomycin twice a day and 500 Mgr of of tetracyline four times a day to all pregnant women had a temperature 98,5 Farenhite or more, or in whom the foetus had a heart rate of more than 160. They found a marked decrease in mortality and in intrauterine pneumonia.

Silverman believed that preventive treatment in premature babies in cases of chorioamnionitis was useful and decresed the mortality. Routine antibiotic chemotherapy was considered, but because of the risk of producing bacterial resistance it was not accepted.

Today the use of wide spectrum antibiotics in the special circumstances mentioned in maternal temperature and increase of foetal heart rate above 160 for more than ten minutes is advised.

Thus, perfect hygiene of the pregnant women, reduction of internal manipulations, prevention of prolonged labour, acceleration of labour is the more important which should be considered. one should try to terminate labour within 24 hours. If an obstetric operation becomes necessary one must remember that foetal resistance will decrease because of infection and the operative risk will increase.

Risk of amniotic infection in Iran is peculiary a diseaee of the poor people who do not understand the importantce of hygiene. This is made clear by considering the cause of death in the 150 autopsies of still and newborn infants which were performed during the year 1962 in the pat_hological laboratory of the women,s Hospital, Tehran.

These postmortem examination were done fairly routinely. The aut_hor tried to examine all neonetal infants who died and stillborn infatns of more than 6 months gestational age, however some cases were not examined.

The first table shows the cause of death in the 150 autopsies carried out:

CAUSE	NUMBER	PERCENAGE
Infection	28	32%
Labour Complication	33	22%
Malformation	21	14%
Maternal disease	5	3 %
Umbilical Hemorrhage	1	60%
Prematurity	11	7%
Hyaline membrane	11	7%
Rh iso_immunization	5	3,5%
Other	6	4%
Unkown	9	6%

born ALIVE 21			
Intrauterine origin			
28	18%		
Neanatal			
21	14%		
Abruptio Placentae		11	
Amniotic Aspiration		5	
Cord Prolapse		5	
Anoxic and traumatic Hemorrhage		12	

If one separates neonatal deaths from stillbirths and considers the cause of death in each group one obtains the following results: There were 104 neonatal deaths and the cause of death was as follows :

Table 2

CAUSE	NUMBER	PERCENTAGE		
Infection	42	Intrauterine origin	21	20 %
		40 % Neanatal	21	20 %

Labour complication	16	15 %
Malformation	11	10 %
Hyaline membrane	11	10 %
Prematurity	11	10 %
Rh iso_immunization	3	3 %
Other	10	10 %

There were 49 stillbirths and the cause of death was as follows:

TABLE

Cause	Number	Percentage
Infection	7	14 %
Labour complication	17	32 %
Malformation	9	17 %
Rh iso_immuniztion	2	4 %
Maternal disease	5	10,5 %
Unknown	6	12,5 %

Two important things should be mentioned:

1_ In all the cases who died from infection with intrauterine origin, the clinical history and pathological findings are compatible with ascending amniotic infection.

2_ The placenta was not received usually for examination. Possibly the author could have identified the cause of death by placental exami_nation.

Comment: Study of the above tables reveals that infection is the most important and more frequent cause of death.

In the Chicago Lyinin hospital of 526 perinatal deaths 21 (3%)were due to infection, and the following table shows the cause of death in the Children,s Hospital of Boston 3_4_18 (Benirschke)

Table

Newborn	327		Stillborm	224	
Hyaline membrane	134	40%	Placenta	84	38%
Infection(pneumonia	57	22%	Infection	12	9%
Other	128	38%	Other	127	56%

Comparison of these percentages with the author's reveals that inf_ection was responsible for 4% of the perneatal deaths in the Chicago in hospital and 32% in the Women's Hospital. The rate of infection is 40% and 14% among the Women,s Hospital's newborn and stillborn respectively and that of Bonirschke is 21% and 6% respectively. In other

words, infection is one of the most important causes of perinatal mortality in the Women's hospital:

It suggests that more care should be taken of the pregnant women's and new born hygiene

Summary : Infection is an important cause of perinatal death in Iran. It can occur in the antepartum, intrapartum and postpartum periods. Infection can reach the foetus by the following routes: 1- Hematogenous spread via maternal blood 2— Ascending amniotic infection 3- transdecidual spread 4— Via fallopian tubes. The two promier' namely, hematogenous and ascending amniotic are the more common and, from a practical point of view, the most important.

The hematogenous route provides a way in which all microorganisms, namely viruses, bacteria, fungi and protozoa, can infect the foetus throughout the whole pregnancy period. Ascending amniotic infoction is more common and occurs usually during labour. In this vaginal bacteria reach the ammiotic cavity directly via the cervical canal. This occurs mostly in labour which has lasted more than 12 hours even when the membranes are still intact or when the membranes have been ruptured for more than 9 hours.

The fetus is generally infected directly through the respiratory or gastrointestinal tract the more severe and common lesion being seen in the lungs (intrauterine pneumonia).

All inflammatory reaction which is seen in the membranes. umbilical cord,placental tissue and foetus is called the «Amniotic infection syndrome» Amniotic infection in severe ends in stillbirth but in the less severe it produces inflammatory symptoms in the first few days after delivery, which are usually the result of intrauteine pneumonia. It is the most important cause of perinatal death. Because of the risk of producing bacterial resistance routine antibiotic and chemo-therapy is not accepted but the use of a wide spectrum antibiotic is advised in special circumstances viz: rise of maternal temperature and increase of foetal heart rate above 160 beet in minute. Thus, perfect hygiene of pregnant women, reduction of internal manipulations, prevention of prolonged labour, acceleration of labour in the presence of ruptured membranes and induction of labour in the primigravida to reduce the lengh of labour are the most important points which should be considered.

The amniotic infection syndroms is an important condition which sould be considered by the obstetrician and. paediatricican.

History of labour is helpful and examination of frozen sections from the umbilical cord is a safe tool for early diagnosis. Culture of the newborn's nasopharyngeal, secretion, identification of the microorganism, performance of an antibiogram and immediate and accurate treatment could possibly save many newborn's lives. Finally the cause of death in 150 autopises which have been carried out durin: a year in the pathologioal laboratory of the Women's Hospital have been analysed. Infection was responsible for 32% of perinatal deaths. The rate of infection was 40 % among the neonatal deaths and 14 % among the stillbirths Half of the neonatal, infection was of intrauterine origin (amniotic type) .

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RESUME

Les infections du foetus et du nouveau né consitutent l'une des principales causes de la mortalité infantelle en Iran. La contamination peut avoir lieu avant la naissance, au corus de la naissance ou bien juste après la naiesance. Les différentes voies de contamination sont les suivantes:

1. Voie sanguine maternelle.
2. Voie ascendante amniotique
3. Voie transdéciduale
4. Voie tubaire.

Parmi ces differentes modes de contamination les deux premières sont les plus importantes et les plus communes

Par Voie sanguine maternelle' toute bactéries, virus, protozoaires ou champignons pathogènes peuvent contaminer le foetus et cette contamination peut avoir lieu dans toute la durée de la grossesse.

Par voie amniotique ascendante la contamination peut avoir lieu au cours de l'accouchement De cette focon les infections vaginales se propagent, par le col uterin, a la cavitè amniotique. Cet incident est beaucopu plus fréquént dans les cas où l'accouchement est de longue durée (plus

de 12h.) ou bien dans les cas ou il y a une rupture précoce des membranes.

L'infection foetale se localise généralement au niveau des voies respiratoires ou gastro-intestinales.

Le processus inflammatoire peut être facilement observé au niveau du cordon ombelical, placenta et les membranes, ce qu'on appele «Syndrome de l'infection amniotique».

Cette infection, dans les cas graves, se termine par la mort prénatale et dans lescas moins sevère le nouveau-né souffre, dès la naissance, d'un état infectieux, qui se termine souvent à la mort. Dans les cas où l,état général de la femme enceinte montre une infectique avec augmentation du nombre de battement du coeur de foetus (pules de 150 par min.) on peut adminstrer les antibiotiques a spectre lage.

A tite de prévention il faut prendre des soins d'hygiène génital chez les femmes enceinte et s'abstenir surtout de toucher vaginal. Dans les cas oû le sac amniotique est dèchiré, tl faut provoquer l'accouchement.

En ce qui concerne le diagnostic, liétat clinique de la femme. la dwrée er la facon de l'accouchement et enfin l,examen extemporané du cordon omb lical peuvent éclairier le diagnostic.

La culture des secretions nasopharyngées du neuveau-né et l'antibiogramme des souches microbiennes isolées sont recommandées.

Au cours d'une année nous avions l'occasion de pratiquer 150 Autopsies du nouveau-né au service d'Anatomie Pathologique de l'Hopital des femme de Téhéran.

L'infection était la cause principale de la mortalité et sa distribution est la suivante:

32% de l'infection prénatale

40% de l'infection néonatale

14% de mort-nés par infection.

Dans la moitié des cas de l'infection néonatale la contamination d'origine intra-uteriue.

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Results of Experiments on Replacement of Superior Vena Cava

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A. Material and Method

Twenty six dogs of both sex were subjects of these experiments. After anesthetising with Nembutal, one grain per lb. body weight, the right hemithorax was opened and superior Vena Cava was exposed. Two internal thorasi veins and one posterior branch were ligated. A piece of vein, one to two inches long, was resected. The replacement was made either with vein itself or with a piece of Edward Tape Nylon or operation. In seven dogs an A. V. shunt was produced in neck between right carotid artery and right Jugular vein in two others anticoagulants were used.

Varieties and Results.

I– Autogenous vein graft.

In seven dogs, autogenous vein graft was used i. e. the vein was resutured. In five directly between distal and proximal end and in other two between distal end right auricle.

Both dogs of second group died within twenty four hours after operation with clotted graft.

Three of the five dogs which had graft between both ends of cava died. One with tension pneumothorax and the other from bleeding. The third dog found dead in her cage on the fifth post operative day, with no cause of death detectable in autopsy. In all these three, the grafts were patent.

Of the remaining two, who survived operation, angiogram showed that only one had patent graft, this dog was sacrificed after five months, the graft was patent with a good epithelial lining. A marked stricture was detected in lower end.

The other dog who survived with a thrombosod graft was also sacrificed five months postop. Autopsy confirmed that the graft is clotted and only a good collateral circulation brings back the blood from head and arms.